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CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP) FOLLOWING CARDIAC SURGERY IN CHILDREN : EFFECTS ON LUNG MECHANICS AND BLOOD GASES

THESE

présentée à la faculté de médecine de l'Université de Genève
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par

Louis Olivier TISSOT DAGUETTE

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La Faculté de médecine, sur le préavis du professeur Pierre Ferrier, autorise l'impression de la présente thèse, sans prétendre par là émettre d'opinion sur les propositions qui y sont énoncées.

Genève, le 30 avril 1979

Le doyen :
André Cruchaud

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A MES CHERS PARENTS,

A JACQUELINE

SUMMARY

The effects of CPAP on lung mechanics, ventilation and blood gases were studied in 14 patients following cardiac surgery for various heart diseases. In addition, resulting changes in cardiac index were estimated. Beneficial effects on lung compliance (C_L) and total lung resistance (R_{TL}) were found in 9 cases, and a reduction of the total work of breathing (W_T) was observed. $Pa0_2$ was significantly improved with increasing levels of CPAP. At a prefixed maximum value of CPAP (15 cm H_20), no deleterious effects on hemodynamics were found.

ABREVIATIONS USED

avD_{O2}	arteriovenous oxygen content difference
C_L	lung compliance
CPAP	continuous positive airway pressure
CPPV	continuous positive pressure ventilation
CVP	central venous pressure
FRC	functional residual capacity
PaC_{O2}	arterial carbon dioxide tension
Pa_{O2}	arterial oxygen tension
R_{AW}	airway resistance
R_{TL}	total lung resistance
$\dot{Q}_T$	cardiac output
$\dot{Q}_I$	cardiac index
$\dot{V}_E$	minute ventilation
V_T	tidal volume
W_T	total work of breathing

Pulmonary dysfunction is frequently observed following cardiac surgery with cardiopulmonary bypass when controlled ventilation is discontinued (13, 39). This disturbance is mainly due to a reduction of functional residual capacity (FRC), resulting in a decreased lung compliance, increased right to left shunt through the lung and increased pulmonary work, leading to hypoxia and respiratory insufficiency respectively (17, 33). Since 1971, continuous positive airway pressure breathing (CPAP) or continuous positive pressure ventilation (CPPV) have been generally accepted as significant advances in the treatment of the newborn with respiratory distress syndrome (18, 21, 43), and for the management of infants and children after open heart surgery (7, 11, 17, 20, 31, 39). By increasing FRC, CPAP and CPPV improve alveolar air mixing and reduce intrapulmonary shunts by recruitment of collapsed perfused alveoli. This in turn increases lung compliance (C_L) (1, 8, 10, 15, 17, 23, 33, 41). Beneficial effects of CPAP or CPPV are related to an optimal level of applied pressure to be determined in each situation (41). Too high a level of CPAP may result in adverse effects, such as a decrease in C_L by overdistension of the alveoli (41), thus increasing the risk of pneumothorax (5, 19, 23, 40, 44). Cardiac output may also be decreased due to diminished venous return (9, 24, 35), direct effects on pulmonary capillaries (22, 34) and left ventricular dysfunction (25, 34).

The aim of the present study was to measure the effect of different levels of CPAP on lung mechanics and arterial blood gases in children who have undergone cardiovascular surgery for various congenital and acquired cardiopathies.

MATERIAL AND METHODS

Fourteen patients recovering from surgery involving cardiopulmonary bypass were studied. Clinical data are given in table I.

After surgery, all patients received assisted ventilation through a nasotracheal tube. When weaning from the respirator was tolerated, blood gases and lung mechanics were studied in each patient at barometric pressure and under different levels of CPAP with unchanged inspired oxygen concentration (Fig. 1). The use of CPAP was not required on clinical basis in any of the children studied, as adequate oxygenation could be achieved without it. Measurements were obtained at steady state 30 minutes after application of each level of CPAP.

Patients sedated by Chlorprothixen (1 mg/kg) were studied in the semi-recumbent position with the trunk elevated approximatively 20 degrees from the horizontal plane. Arterial blood samples were drawn from an indwelling radial artery catheter and duplicate blood gases were performed immediately (IL Blood Gas Analyser 213). Mean arterial blood pressure was continuously measured (Statham P23 Db with a Philips amplifier). A catheter positioned in the right atrium enabled sampling of venous blood and measurement of central venous pressure (CVP). Arterial venous differences in calculated oxygen content (av DO_2) were used to estimate effects of CPAP on cardiac index ($\dot{Q}_I$) (12, 41).

The technique used for measuring dynamic lung compliance and total lung resistance (R_{TL}) is that of Mead and Whittenberger (27), adapted for infants by Polgar and Lacourt (32). Briefly, the basic principles are as follows. Three variables are necessary to determine these two parameters : a) Measurement of flow is obtained by a screen-mesh pneumotachograph mounted in series between the nasotracheal tube and the CPAP apparatus (Fig. 1) ; b) Tidal volume (V_T) is derived by

integration of the flow signal through the respiratory cycle ; c) Changes in transpulmonary pressure necessary to overcome the resistive and elastic forces are transmitted from a thin latex balloon (diameter 5 mm/length 6-7 cm) filled with 0,5 ml of air and connected to a strain gauge system by a catheter (diameter 2 mm) (26, 27, 28) passed through the nose. The balloon is placed at the junction of upper and middle thirds of the oesophagus. Pressure changes are simultaneously plotted on the X axis of an X-Y oscilloscope, with the flow or volume signal (on the Y-axis) to measure R_{TL} or C_L respectively. The slope of the loop obtained is measured with a protractor, after electronical substraction of a fraction of pressure proportional to volume, when measuring resistance and substraction of a fraction of pressure proportional to flow when measuring compliance. Twenty sets of measurements were made for each level of CPAP and the average values were calculated.

Work of breathing (W_T) was calculated by substituting the determined elastic and resistive factors, V_T and respiratory rate (f) in the modified formula of Otis, Fenn and Rahn (30) :

$$W \text{ (g. cm min}^{-1}) = \tfrac{1}{2} f K_{el} (V_T)^2 + \tfrac{1}{4} K_r \overline{TI}^2 f^2 (V_T)^2$$

- where $K_{el} = \frac{1}{C_L}$ with C_L expressed in ml/cm H_2O

- and $K_r = R_{TL}$ in cm H_2O per ml/minute.

The protocol was approved by the Ethical Committee of the Department of Pediatrics. The progressive increases in pressure as well as the abrupt weaning at the end of the study were well tolerated by all patients. Statistical analysis was performed using the Student test for paired observations (38).

RESULTS

Individual results of lung mechanics, hemodynamics and

blood gases at each level of CPAP are shown in table II and mean values in table III. Since in normal patients, C_L, R_{TL} and V_T vary with age and weight, the changes in these parameters are also expressed as a percentage of the initial value.

Effects of CPAP on lung mechanics

Table III and Fig. 2 show that there was a significant increase in mean value of C_L with increasing levels of CPAP. The values of C_L obtained at 15 cm H_2O showed a tendency to remain elevated for at least 30 minutes after CPAP was discontinued. We were unable to demonstrate a significantly different pattern of changes in C_L between the various cardiopathies studied. However, in three individual cases, two with mitral disease and one with atrio-ventricular canal defect, C_L decreased with increasing levels of CPAP. Vibrations in the system prevented us from measuring R_{TL} at each level of pressure in every patient and thus we were unable to calculate statistical differences. However, figure 3 suggests a decreasing trend in R_{TL} as CPAP increases. In the 12 cases in which both C_L and R_{TL} were measured, changes (as expressed in per cent for both parameters) were in opposite directions in 10 instances (Fig. 4). Decrease in C_L was accompanied by a decrease in R_{TL} in 2 instances (cases 10 and 14) and an increase in R_{TL} in one patient (case 12).

Calculated total work (W_T) showed an obvious decrease in 8 cases where C_L increased and R_{TL} decreased. In case 12, there was a large W_T increase following a decrease of C_L and an increase of R_{TL}. This expected correlation was not found in 3 patients (cases 6, 10, 14).

Since there was no significant difference in respiratory rate, the increase in minute ventilation ($\dot{V}_E$) was due to a significant increase

in V_T (Table III and Fig. 2). As for C_L, there was no significant change in V_T when CPAP was stopped.

Effects of CPAP on hemodynamics

Changes in pulse rate, mean arterial pressure and oxygen content differences, as indices of cardiac output were not significant throughout the experiment (Table III). Central venous pressure, however, increased linearly and significantly with increases in CPAP and fell to initial values when the patients were returned to atmospheric breathing.

Effects of CPAP on blood gases

The Pa_{O_2} rose significantly with increasing levels of CPAP. When no more pressure was applied, there was a tendency for Pa_{O_2} to remain elevated. There was an inverse and significant relationship for PaC_{O_2} (Table III and Fig. 2). The pH remained essentially stable throughout the study.

DISCUSSION

In this study we have demonstrated that dynamic compliance increases with increasing CPAP (Fig. 2, and Table III). This beneficial effect has been shown to be due to improvement in FRC by recruitment of collapsed alveoli (10, 17, 31, 33) and to prevention of units of lung from reaching their closing volume (10). During CPPV, resorption of interstitial fluid into the circulation has also been postulated (1, 23). From the experience of others using CPPV (29, 41) or CPAP (43), one expects that too high a pressure decreases C_L since the lung reaches the limit of its elasticity (2). For safety reasons, we limited our study to a maximal pressure of 15 cm of H_2O, and our data therefore

fail to show at which level of pressure this effect occurs. Previous volume history of the lung explains the finding that C_L for the whole group did not return to initial values when CPAP was discontinued for at least twenty minutes (36). Except for one infant with tetralogy of Fallot (case 2) in which the CPAP resulted in the highest C_L at 10 cm H_2O, the greatest positive change in C_L was measured at the highest applied level of CPAP. Thus the best CPAP could not be demonstrated. In three children (cases 10, 12, 14), C_L diminished when CPAP was applied. The greatest negative change was observed in a patient with mitral disease (case 14), in whom a high initial C_L was measured.

As for C_L, increases in thoracic gas volume play an important role in lowering a component of R_{TL} ; that is, airway resistance (R_{AW}) (14). This explains the parallel changes, but in opposite direction in ten of our patients (Fig. 4). However, when the applied pressure was decreased to zero, the abrupt return of R_{TL} to its initial value in the presence of a persistently high C_L, indicates that CPAP acts mostly on resistance by a volume independent mechanism. In infants and children who have very compliant airways, the positive applied pressure decreases R_{AW} by dilatating the airways (7, 37). In two children (cases 10 and 14), a decrease in R_{TL} was observed in the presence of a diminished C_L, lending support to the importance of this direct mechanical factor.

In eight patients in whom the significant changes in C_L and R_{TL} were found in opposite direction, W_T also decreased as expected. This beneficial effect of CPAP on C_L and R_{TL} was also demonstrated in patient n° 6, but resulted in an increased W_T secondary to a large increase in $\dot{V}_E$. The marked decrease in R_{TL} observed in patient n° 10 may partially explain the diminished W_T, despite the decrease in C_L. Adverse effects of CPAP were found in a patient with mitral disease (case 12), resulting in decreased C_L, increased R_{TL} and W_T. The

clinical condition of this child improved when the applied pressure was withdrawn.

Several authors (22, 25, 34, 41) have shown that an excessive increase in airway pressure can produce a decrease in $\dot{Q}_T$ and thus diminish local oxygen supply to the tissues. Since the level of CPAP required to produce optimal oxygenation without interference with $\dot{Q}_T$ is lung compliance dependent (4), the lack of significant change in pulse rate mean arterial pressure and in $\dot{Q}_T$ (as estimated by $avDO_2$) reported in this study indicate that we did not reach the level of airway pressure at which this would occur. The finding that patients n° 10, 12 and 14 were hemodynamically stable despite a decrease in C_L of 40 % suggests that only very important changes in C_L may alter $\dot{Q}_T$ (Fig. 4). The rise in CVP which follows the use of CPAP may protect against a change in $\dot{Q}_T$ by maintaining an effective right ventricular filling pressure (22). Close control of blood volume throughout the study may have prevented significant changes in the hemodynamics of our patients (35, 42, 45).

As in previous studies (3, 7, 11, 17, 18, 20, 21, 43), we found that CPAP has beneficial effects on blood gases. The improved mean PaO_2 found for the group when CPAP is at its higher level (Fig. 2) can be explained by an increase in FRC, $\dot{V}_E$ and by suppression of right to left shunt secondary to a low ventilation perfusion ratio. Interstitial water shifts favored by positive end expiratory pressure may play a role in increasing PaO_2 by improving diffusion (1, 23). There is, however, no universal agreement on this effect (6, 29). The decrease in Pa_{O2} following application of CPAP, which occurred in case n° 5 and 14, may be explained by an increase in right to left pulmonary shunt due to an unexpected redistribution of blood flow

through unventilated lung areas (33).

Increases in $\dot{V}_E$ secondary to a larger V_T improves alveolar ventilation, thus lowering Pa_{CO_2} for the whole group. This result diverges from previously reported findings (2, 7, 20, 43) but is in accordance with those of Flenley et al (16).
A decrease in the alveolar dead space/V_T ratio secondary to increases in C_L, as reported by Suter et al (41), may also play a role in the decrease of Pa_{CO_2} observed in our patients.

CONCLUSION

CPAP effectively supports ventilation of children after cardiovascular surgery by improving pulmonary mechanics and arterial oxygenation. At the prefixed maximum value of CPAP (15 cm H_2O), the best CPAP could not be demonstrated.

TABLE I

CLINICAL DATA

Case n°	Age (years and months)	Weight (Kg)	Height (cm)	Diagnosis	Operation	Time interval after operation (hours)
1	3 9/12	13,8	98	TF	Total correction	24
2	1	7,6	73	TF	" "	24
3	1 6/12	12	85	TF	" "	4
4	3 1/12	14	101	TF	" "	48
5	3 4/12	15	97	VSD + AR	" "	24
6	1	7,1	75	d - TGV	Senning	24
7	7/12	5	66	d - TGV	"	168
8	7/12	5	66	d - TGV	"	60
9	1 11/12	11,5	82	VSD	Total correction	20
10	4 5/12	12	97	AVC	" "	24
11	12	30	134	MS + MR	Starr	24
12	12	19,5	125	MS + MR	"	24
13	5	17,3	108	MS + MR	Hancock	24
14	16	30	140	MS + MR	Starr	21

TF : Tetralogy of Fallot.

AR : Aortic regurgitation.

VSD : Ventricular septal defect.

TGV : Transposition of the great vessels.

AVC : atrio-ventricular canal defect.

MS : Mitral stenosis.

MR : Mitral regurgitation.

TABLE II (a and b)

INDIVIDUAL RESULTS OF LUNG MECHANICS AND BLOOD GASES AT EACH LEVEL OF CPAP

Patients	CPAP cmH_2O	C_L ml/cmH_2O	R_{TL} $cmH_2O/l/s$	Respiratory rate breaths/min	V_T ml	W_T g/cm/min	$avDO_2$ Vol %	Mean arter. Blood pres. kPa	CVP cmH_2O	Heart rate beats/min	PaO_2 kPa	$PaCO_2$ kPa	ph
	0	-	-	-	-	-	6,05	-	-	-	7	5,2	7,38
1	5	5,5	50	60	66	55965	6,15	11,7	9	130	9,7	4,9	7,39
	10	5,5	35	48	65	32429	6,14	12,2	9	125	11,4	4,6	7,41
	15	9,5	30	46	70	24641	7,01	12,2	11	120	14,1	4,1	7,42
	0	5	60	50	60	40184	5,55	11,7	8	120	10,0	4,1	7,39
	0	10	6	60	50	9718	3,7	8,0	11	160	9,3	3,0	7,36
2	5	12	-	60	70	-	3,45	8,0	11	160	11,4	2,9	7,42
	10	15	4	42	45	3419	2,41	8,4	12	165	13,2	3,2	7,45
	15	11,5	-	56	60	-	3,08	7,4	13	155	13,2	2,9	7,40
	0	13	-	52	70	-	2,49	9,3	10	160	8,8	3,6	7,39
	0	8	-	44	62	-	8,76	10,0	10	140	15,7	3,6	7,40
3	5	10	-	40	80	-	8,25	9,8	10	140	18,2	3,5	7,42
	10	20	-	36	86	-	8,18	10,0	16	145	17,1	2,8	7,41
	15	45	-	34	96	-	8,70	10,6	19	140	19,7	3,7	7,42
	0	90	-	-	120	-	10,01	10,6	13	130	18,9	3,0	7,41
	0	22,5	8	28	230	46531	4,33	9,3	17	135	9,8	4,4	7,41
4	5	28	6	32	250	51489	5,31	10,4	15	125	16,0	4,4	7,43
	10	25	8	32	200	39048	4,88	10,6	16	126	17,4	3,9	7,42
	15	38,5	6,5	25	200	19642	5,31	11,3	-	135	17,6	3,5	7,41
	0	-	9	38	140	-	5,58	10,9	15	124	17,3	3,6	7,43
	0	28	8	28	100	7574	3,76	10,9	13,5	125	14,9	4,3	7,40
5	5	40	6	26	110	5948	3,19	10,4	14	126	14,1	3,5	7,39
	10	44	-	32	100	-	3,17	10,6	16	124	10,8	3,3	7,37
	15	60	-	32	100	-	1,54	10,6	17	124	8,0	3,9	7,40
	0	50	-	28	100	-	2,98	11,2	13	120	16,3	2,9	7,42
	0	9,5	-	28	100	-	-	9,0	13	132	12,5	4,0	7,42
6	5	12,5	20	29	120	26650	-	8,0	12	124	12,5	3,7	7,41
	10	18	14	32	140	28958	-	8,0	14	120	13,9	3,7	7,41
	15	17	14	30	150	31491	-	8,0	-	130	12,4	3,2	7,42
	0	16	-	32	140	-	-	9,0	11	112	11,4	3,2	7,42
	0	4	30	52	38	14823	-	7,4	-	88	11,4	5,2	7,44
7	5	10	20	44	40	6064	-	7,4	-	80	12,5	5,3	7,44
	10	7	16	44	48	10170	-	8,5	-	90	13,3	4,1	7,48
	15	13,5	16	45	62	11517	-	10,0	-	110	12,6	4,5	7,44
	0	-	-	-	-	-	-	-	-	-	-	-	-

TABLE II (b)

Patients	CPAP cmH_2O	C_L ml/cmH_2O	R_{TL} $cmH_2O/l/s$	Respiratory rate breaths/min	V_T ml	W_T g/cm/min	$avDO_2$ Vol %	Mean arter. Blood pres. kPa	CVP cmH_2O	Heart rate beats/min	PaO_2 kPa	$PaCO_2$ kPa	ph
	0	10	36	52	62	25366	3,54	8,0	-	132	10,9	3,7	7,34
8	5	12,5	20	46	75	20125	2,62	8,2	-	132	14,8	3,7	7,40
	10	11	16	44	76	18637	3,17	8,2	-	128	16,5	3,9	7,35
	15	20	12	50	84	17516	2,16	8,2	-	132	17,0	4,0	7,35
	0	13,5	28	65	72	65120	3,40	9,0	-	132	12,1	3,3	7,43
	0	19	32	54	100	52313	3,93	10,6	16,5	146	14,4	4,8	7,32
9	5	25,5	26	42	195	101813	4,71	10,1	15,5	124	14,4	4,1	7,33
	10	37,5	14	36	130	21068	5,07	10,0	15	112	14,6	3,9	7,34
	15	42,5	8	36	180	27508	-	10,0	16	116	16,1	4,5	7,35
	0	21	24	52	96	35846	5,00	10,0	12,5	116	16,7	4,1	7,35
	0	-	-	-	-	-	-	-	-	-	-	-	-
10	5	24	16	30	125	18879	3,80	10,6	13,5	90	19,9	4,4	7,49
	10	18	10	30	100	12024	3,82	12,0	14,5	95	21,0	4,4	7,51
	15	15	6	32	87,5	10098	3,90	10,6	16	95	19,3	4,5	7,51
	0	40	13,5	32	100	9674	4,10	10,6	11,5	105	20,3	5,0	7,46
	0	35	-	28	125	-	10,15	9,3	11	126	11,4	5,0	7,42
11	5	43	-	20	150	-	8,16	10,1	12	118	12,0	4,9	7,42
	10	46	-	20	190	-	7,50	9,4	15	116	12,4	5,6	7,40
	15	55	-	18	210	-	6,93	9,8	16	124	13,7	5,0	7,42
	0	45	-	14	-	-	8,67	-	-	-	12,4	4,2	7,42
	0	25	16	24	125	13409	7,80	11,7	11	100	21,3	4,9	7,43
12	5	20	18	24	160	26263	6,10	11,7	11	104	19,9	5,0	7,43
	10	17	20	16	175	20850	7,20	11,8	12	98	21,3	4,9	7,46
	15	20	16	20	200	30506	7,21	12,0	15	104	21,9	5,1	7,44
	0	25	20	22	175	25648	8,00	11,7	12	104	22,5	4,9	7,47
	0	60	8	24	275	29426	3,44	9,0	5	155	14,2	4,9	7,49
13	5	50	8	26	275	36446	2,62	9,0	9	155	24,7	7,2	7,47
	10	90	6	32	250	26886	4,20	8,5	10	150	25,1	4,1	7,40
	15	120	-	24	350	-	4,33	8,5	10	156	31,6	4,8	7,39
	0	107	-	28	375	-	4,51	10,4	10	150	31,6	4,4	7,44
	0	-	-	32	250	-	3,95	10,6	8	108	11,6	4,9	7,45
14	5	140	4	36	250	21292	3,82	10,1	10	120	10,2	5,2	7,41
	10	110	4	32	250	18555	3,33	10,6	12	110	10,2	4,8	7,47
	15	80	-	40	300	-	-	-	-	-	-	-	-
	0	-	-	-	-	-	-	-	-	-	-	-	-

TABLE III

EFFECTS OF CPAP ON LUNG FUNCTION, HEMODYNAMIC AND BLOOD GASES

(Mean ± standard error of the mean)

	Mean initial value	Change from mean initial value (in per cent) and Mean value			
End expiratory pressure (cmH_2O)	0	5	10	15	0
Dynamic compliance ml/cmH_2O	21 ± 4,9	+ 47 30,9 ± 9,1	+ 57,6 * 33,1 ± 8,3 *	+ 86,2 * 39,1 ± 8,5 *	+ 84,3 38,7 ± 10
V_t (ml)	122 ± 20,4	+ 14,7 * 140 ± 20,4 *	+ 8,6 132,5 ± 18,8	+ 25,8 * 153,5 ± 24,5 *	+ 7,9 131,6 ± 26,5
Heart rate (beats/min)	128,9 ± 6,2	- 4,3 123,4 ± 5,8	- 5,6 121,7 ± 5,7	- 2,1 126,2 ± 5	- 3,2 124,8 ± 5,3
Mean arterial blood pressure (kPa)	9,48 ± 0,38	+ 2,1 9,68 ± 0,36	+ 4,6 9,92 ± 0,38	+ 4,9 9,95 ± 0,42	+ 9,7 * 10,4 ± 0,29 *
$avDO_2$ vol. %	5,4 ± 0,7	- 10,4 4,84 ± 0,6	- 8,9 4,92 ± 0,6	- 7,2 5,01 ± 0,8	+ 1,8 5,5 ± 0,7
PaO_2 (kPa)	12,6 ± 1	+ 19 15 ± 1,1	+ 23 * 15,5 ± 1,2 *	+ 32,5 * 16,7 ± 1,6 *	+ 30,9 16,5 ± 1,8
$PaCO_2$ (kPa)	4,4 ± 0,2	+ 1,8 4,48 ± 0,3	- 7 * 4,09 ± 0,2 *	- 6,1 * 4,13 ± 0,2 *	- 12,3 * 3,86 ± 0,2 *

* Significant at 0,05 level from initial values.

Figure 1 : System for applying CPAP and measuring lung function.

Figure 2 : Effects of CPAP on compliance (C_L) and tidal volume (V_T) (in percent change), and on arterial oxygen (Pa_{O_2}) and arterial carbon-dioxide (PaC_{O_2}) tensions.

Figure 3 : Effect of CPAP on total lung resistance (R_{T_L}) (12 patients).

Figure 4 : Maximum changes observed in compliance ($_{CL}$), total lung resistance (R_{TL}) and work of breathing (W_T) to applied CPAP (5, 10 or 15 H_2O) (expressed as percent changes from values obtained at atmospheric pressure).

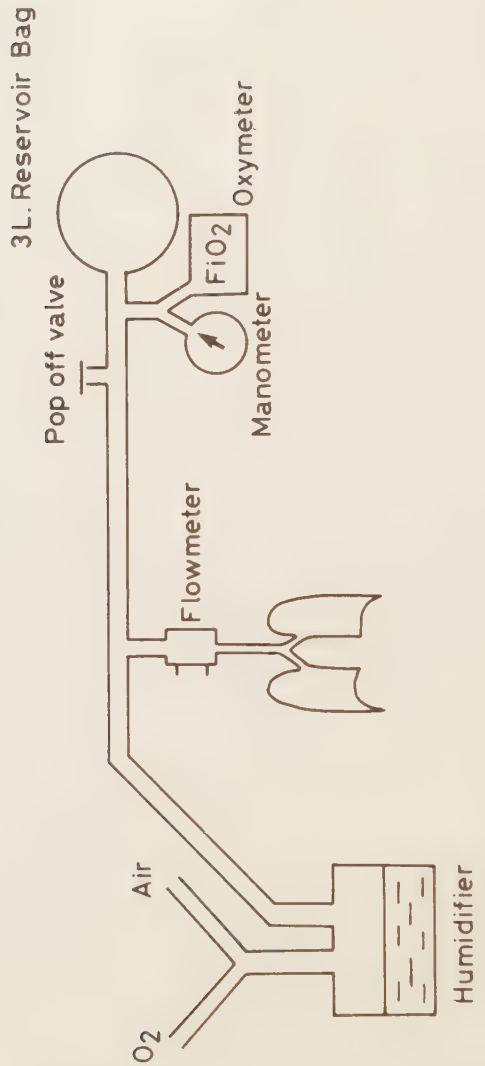
3L. Reservoir Bag
Pop off valve
Oxymeter
FiO_2
Manometer
Flowmeter
Air
O_2
Humidifier

Figure 1

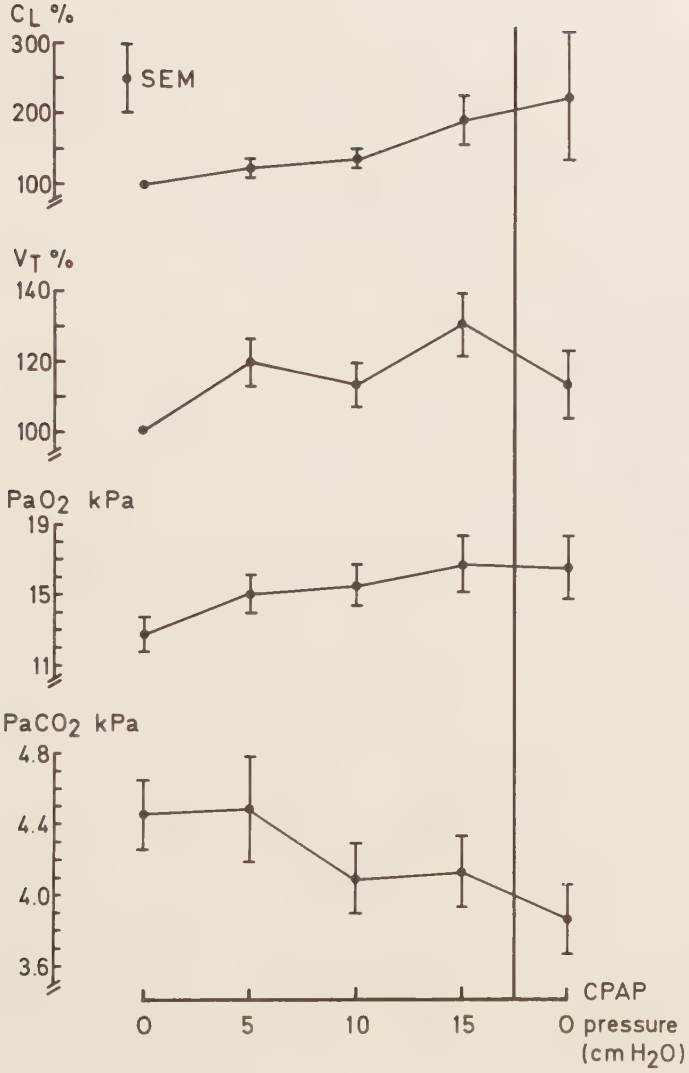
C_L %
300
200
100
SEM
V_T %
140
120
100
PaO_2 kPa
19
15
11
$PaCO_2$ kPa
4.8
4.4
4.0
3.6
CPAP
0
5
10
15
0 pressure
(cm H_2O)

Figure 2

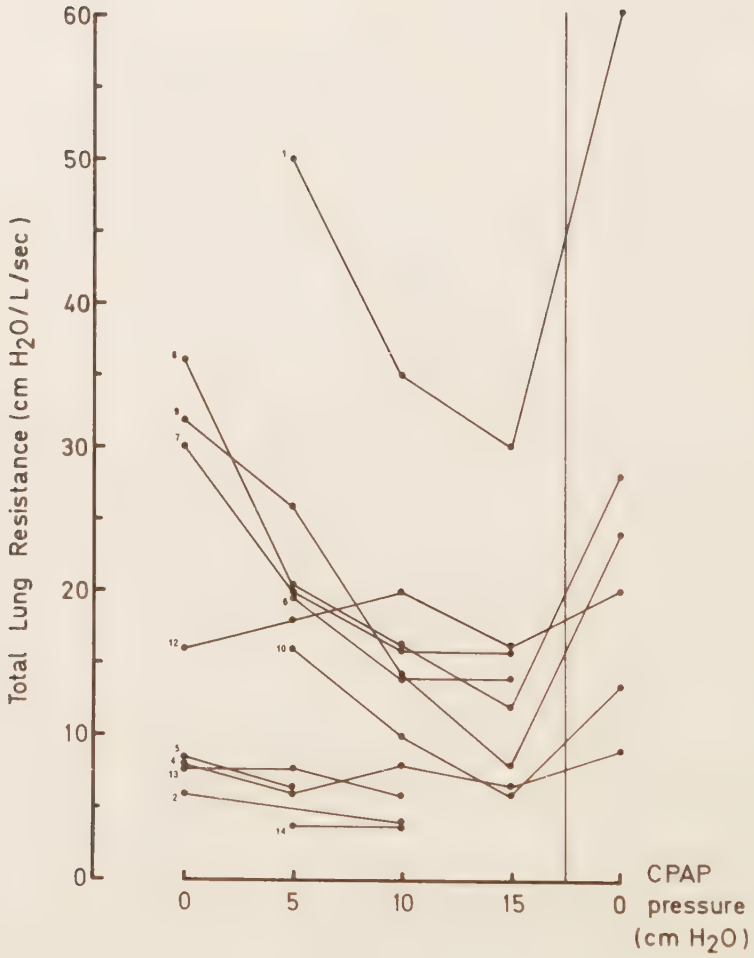
Total Lung Resistance (cm H2O/L/sec)
60
50
40
30
20
10
0
0
5
10
15
0
CPAP pressure (cm H2O)

Figure 3

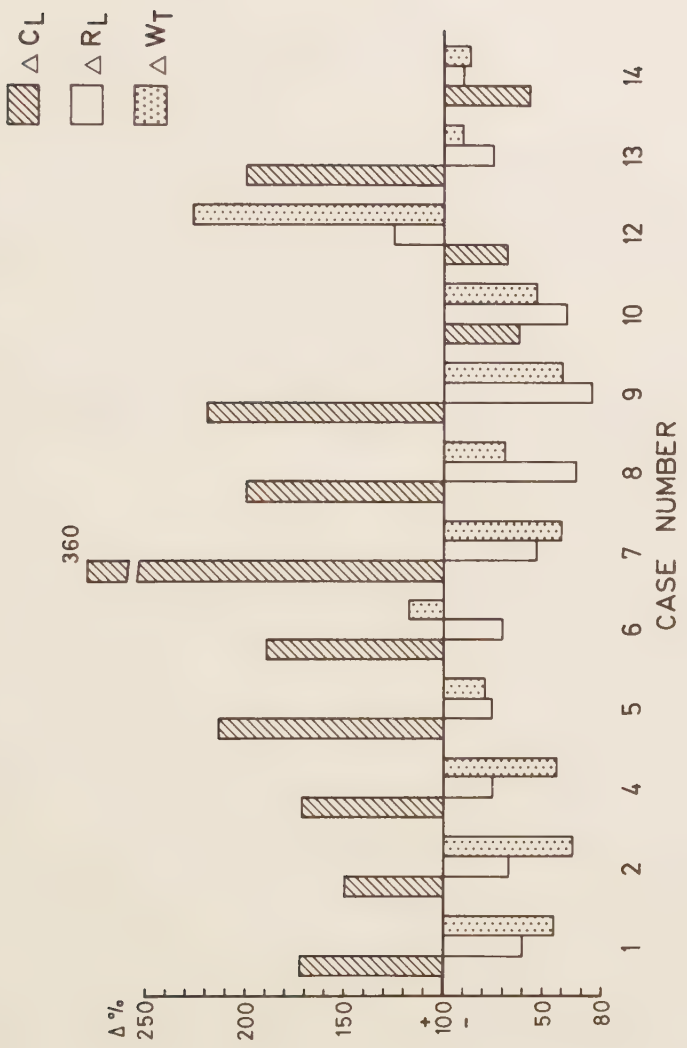
Δ CL
Δ RL
Δ WT
360
Δ %
250
200
150
+100
-
50
80
1
2
4
5
6
7
8
9
10
12
13
14
CASE NUMBER

Figure 4

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